L4 ANSWER 13 OF 19 MEDLINE DUPLICATE 5
AN 96124890 MEDLINE
DN 96124890 PubMed ID: 8548754
TI Tumor necrosis factor-alpha allelic frequency and **chromosome 6** allelic imbalance in patients with **colorectal cancer**.
AU Honchel R; McDonnell S; Schaid D J; Thibodeau S N
CS Department of Laboratory Medicine, Mayo Clinic and Foundation, Rochester, Minnesota 55905, USA.
SO CANCER RESEARCH, (1996 Jan 1) 56 (1) 145-9.
Journal code: 2984705R. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199602
ED Entered STN: 19960306
Last Updated on STN: 19960306
Entered Medline: 19960216
AB The human tumor necrosis factor (TNF) locus is located on **chromosome 6p21**.3 and contains at least five polymorphic microsatellites. In this study, we compared the allelic frequencies derived from 50 normal controls to 64 patients with **colorectal cancer** at one of these loci, TNF alpha. No differences in allelic frequencies were observed between these two groups (P = 0.47). However, sequencing of the TNF alpha PCR product revealed two populations of TNF alpha alleles; alleles with the expected DNA sequence (i.e., the expected number of AC/GT repeats) and alleles that contained 8-bp deletions adjacent to the microsatellite repeat. In **addition**, we also examined paired normal and tumor DNA from the **colorectal cancer** group for microsatellite alterations at the TNF alpha locus, including allelic **loss** of heterozygosity and microsatellite instability. Of the 64 tumors examined, 13 (20%) demonstrated microsatellite instability, and 14 (42%) of 33 informative cases demonstrated allelic imbalance. Analysis of 10 additional **chromosome 6** loci for allelic **loss** showed that 23 (47%) of 49 informative cases exhibited allelic imbalance with at least one **chromosome 6p** marker, 23 (47%) of 49 with at least one 6q marker, and 29 (59%) of 49 with at least one marker on **chromosome 6**. Examination of tumors for the minimal region of **deletion** overlap suggests the presence of tumor suppressor genes on both 6p and 6q.

L4 ANSWER 12 OF 19 CAPLUS COPYRIGHT 2003 ACS
AN 1996:267491 CAPLUS
DN 124:313966
TI Comparative genomic hybridization reveals a specific pattern of chromosomal gains and losses during the genesis of colorectal tumors
AU Ried, Thomas; Knutzen, Regina; Steinbeck, Ruediger; Blegen, Harald; Schroeck, Evelin; Heselmeyer, Kerstin; du Manoir, Stanislas; Auer, Gert
CS National Center for Human Genome Research, NIH, Bethesda, MD, 20892, USA
SO Genes, Chromosomes & Cancer (1996), 15(4), 234-45
CODEN: GCCAES; ISSN: 1045-2257
PB Wiley-Liss
DT Journal
LA English
AB Comparative genomic hybridization was used to screen the DNA extd. from histol. defined tissue sections from consecutive stages of colorectal carcinogenesis for chromosomal aberrations. No aberrations were detected in normal epithelium. **Gain** of chromosome 7 occurred as a single event in low-grade adenomas. In high-grade adenomas, an over-representation of chromosomes 7 and 20 was present in 30% of the cases analyzed. The transition to colon carcinomas was characterized by the emergence of multiple chromosomal aberrations. Chromosomes 1, 13, and 20 and chromosome arms 7p and 8q were frequently gained, whereas **chromosome 4** and chromosome arms 8p and 18q were recurrently underrepresented. The same tissue sections that were used for CGH were analyzed by DNA-ploidy measurements and immunohistochem. staining to quantify proliferative activity and p21/WAF-1 and TP53 expression. The authors obsd. that crude aneuploidy and increased proliferative activity are early events in colorectal carcinogenesis, followed by TP53 overexpression and the acquisition of recurrent chromosomal gains and losses during the progression from high-grade adenomas to invasive carcinomas.

L4 ANSWER 18 OF 19 MEDLINE
AN 92367431 MEDLINE
DN 92367431 PubMed ID: 1502889
TI Oncogenesis in ovarian cancer.
AU Borresen A L
CS Dept. of Genetics, Norwegian Radium Hospital, Oslo.
SO ACTA OBSTETRICIA ET GYNECOLOGICA SCANDINAVICA. SUPPLEMENT, (1992) 155 25-30. Ref: 38
Journal code: 0337655. ISSN: 0300-8835.
CY Sweden
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199209
ED Entered STN: 19920925
Last Updated on STN: 19920925
Entered Medline: 19920917
AB Tumorigenesis is a multistep process involving mutations of dominantly acting proto-oncogenes and mutations and **loss**-of-function mutations of tumor suppressor genes. Some of these mutations may be inherited, but most of them are acquired. Models for the sequential steps of the genetic changes involved in tumor development have been proposed for certain cancers, such as **colon cancer**. In the case of ovarian cancer, relatively little is known about the genetic events associated with the initiation or subsequent progression and metastases of the tumor. Cytogenetic analysis has revealed a high incidence of both structural and numerical chromosome changes, and the extent of these changes seems to increase with tumor progression. Oncogene activations of the proto-oncogenes K-ras, c-myc and c-erbB-2 have been found more frequently in aggressive ovarian tumors and may be associated with poor survival. Tumor-specific allele **loss** involving putative tumor suppressor genes has been observed for loci at chromosomes 11p, 17p, and 17q,--loci commonly deleted in other cancers too. A relatively high incidence of allelic **loss** on **chromosome 6q** appears to be specific to ovarian carcinoma. Familial breast/ovarian cancer has been suggested to map to chromosome 8q. Recently we have found a germ-line mutation in the tumor suppressor gene p53 in a family with breast- and ovarian cancers, indicating that this is the predisposing gene in this family. Genetic changes important for the etiology of ovarian cancers seem to involve both somatic mutations of oncogenes and somatic or germ-line inactivation of tumor suppressor genes.

L4 ANSWER 16 OF 19 MEDLINE
AN 93073667 MEDLINE
DN 93073667 PubMed ID: 1332582
TI A study of **chromosome 6** allele **loss** in human colorectal carcinomas.
AU Wildrick D M; Alfaro S R; Gope R; Boman B M
CS Creighton Cancer Center, Creighton University School of Medicine, Omaha, Nebraska 68178.
SO ANTICANCER RESEARCH, (1992 Sep-Oct) 12 (5) 1717-9.
Journal code: 8102988. ISSN: 0250-7005.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199212
ED Entered STN: 19930122
Last Updated on STN: 19930122
Entered Medline: 19921216
AB Matched normal/tumor DNA pairs from patients with sporadic and hereditary (FAP = familial adenomatous polyposis) **colorectal carcinoma** were examined for tumor-specific allele **loss** on **chromosome 6** using cDNA probes for the avian myeloblastosis viral oncogene homologue (MYB on 6q22-q23), the estrogen receptor (ESR on 6q24-q27), and for the alpha polypeptide of human chorionic gonadotropin (CGA on 6q14-q21). No **chromosome 6** allele **loss** was observed at these gene loci among 22 colorectal carcinomas examined, although such losses were relatively frequent (37.5% of informative individuals) at the D17S28 locus on chromosome 17. These results are consistent with karyological studies and indicate that **chromosome 6** allele **loss** from colorectal carcinomas may occur less frequently than previously reported.